



Comparison on Safety and Efficacy of Telmisartan and Losartan with their Effects on Cardiovascular – A Single Centre Study

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Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

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ABSTRACT

Two of the major determinants of cardiovascular disease, hypertension and hyperlipidemia, commonly coexist. There is high prevalence (5 -25%) of low high density lipoprotein (HDL) cholesterol and a higher prevalence of elevated Triglyceride (TGL) levels in hypertensive compared to normotensive individuals. Elevated total cholesterol (TC) levels augment the risk of cardiovascular disease associated with hypertension. This study aimed to determine the antihypertensive & pleiotropic effects of telmisartan and losartan in patients with mild to moderate hypertension. All the subjects were between the ages of 18 -65 years. 100 subjects were selected for the study and their serum total cholesterol, HDL, LDL, VLDL, Triglyceride, Systolic blood pressure, diastolic blood pressure, heart rate were analyzed at the beginning of the study and 12th and 24 the week. Adverse events are also noted in each visit. All data entered in SPSS 10 version and scores obtained were statistically evaluated using student t test, chi square test and ANOVA. The study showed both telmisartan and losartan good control over hypertension in patients. Telmisartan has pleiotropic positive effect on lipid profile.

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1. INTRODUCTION

Systemic arterial hypertension is a condition that affects almost one billion people worldwide and is a leading cause of morbidity and mortality [1]. This disease is sometimes called the silent killer [2]. The disease in the majority of the cases is asymptomatic until the damaging effect of hypertension, such as stroke, myocardial infarction, renal dysfunction, visual problem are observed. In 90-95% of patients, the cause of hypertension is unknown, which is called primary or essential hypertension. The remaining 5-10% of patients has hypertension that secondarily results from some other disorders such as renal disease, endocrine disease, or other identifiable cause [3].

Two of the major determinants of cardiovascular disease, hypertension and hyperlipidemia, commonly coexist. There is high prevalence (5 - 25%) of low high density lipoprotein (HDL) cholesterol and a higher prevalence of elevated triglyceride (TGL) levels in hypertensive compared to normotensive individuals [4]. Elevated total cholesterol (TC) levels augment the risk of cardiovascular disease associated with hypertension. In fact, a large proportion of the cardiovascular risk in patients with hypertension can be attributed to dyslipidemia. The high attendant cardiovascular risk when these two conditions coexist warrants a strict emphasis on dietary and pharmacological therapy to successfully achieve blood pressure control. Contrary to the goal, it is reported that only 32% of hypertensive patients manage to improve their lipid profile, while this percentage falls to 11% for control of both blood pressure (BP) and lipids [5].

Patients with the common lipid triad (hypertriglyceridemia, high low-density lipoprotein cholesterol [LDL] and low HDL) are at high risk for cardiovascular disease. This risk is even greater when the lipid triad is accompanied by hypertension and diabetes [6].

Hypertension is a term used to describe high blood pressure. Flow of blood is based on the beat of which the heart pumps blood. Hypertension occurs as a result to long duration of abnormal pressure of the main arteries. The term high blood pressure exists in the local Ghanaian Twidialect as "Mogya Mboroso". This literally means overflow of blood and it is the

commonly known and used term for high blood pressure [7].

The renin-angiotensin-aldosterone system (RAAS) is an important mediator in the pathophysiology of hypertension [8]. Evidence also suggests that the RAAS plays an important role in target ions, organ damage, potentially leading to left ventricular hypertrophy (LVH), congestive heart failure (CHF) and end-stage renal disease (ESRD) [9]. The peptide angiotensin II (All) is a primary effector of the RAAS. Angiotensin converting enzyme (ACE) inhibitors block the RAAS by inhibiting conversion of angiotensin I to All, whereas All receptor antagonists, including telmisartan, have the potential to block the system more completely through antagonism of All binding to the All type 1 (AT1) receptor, thereby inhibiting the vasoconstrictor and aldosterone-secreting effects of Angiotensin II [10].

Angiotensin II receptor blockers, a new class of antihypertensive agents, inhibit the renin-angiotensin system by selectively blocking the AT1 subtype of All receptors. Despite sharing this common mechanism of action, pharmacologic differences that could result in different efficacy and tolerability profiles do exist among the AT1 blockers [11]. Losartan, the first orally available AT1 blocker to be developed for clinical use, is a competitive antagonist [12]. Much of the All-inhibiting effect of losartan can be attributed to its active metabolite [13]. Telmisartan is a highly selective, competitive non-peptide AT1 receptor antagonist. Pharmacological effect of All, the primary effector of the renin-angiotensin-aldosterone system [14]. The drug Properties has no affinity for AT2 or other receptors. The antihypertensive drugs in use today were designed primarily to affect cellular and biochemical mechanisms contributing to increased blood pressure, and not to address the disordered lipid metabolism that often accompanies hypertension. Angiotensin II receptor blockers (ARB) are efficient antihypertensive agents that act through inhibition of AT1 receptors. In experimental models, as well as some human studies, ARB has demonstrated the ability to affect lipid metabolism in a modest but significant way [15].

More precisely, ARB improved the overproduction and accumulation of TGL in the liver, in experimental models, through

mechanisms independent of their hypotensive action [16]. Furthermore, there are preclinical studies showing that telmisartan has a beneficial effect on metabolic parameters, including lipid abnormalities, due to its partial activation of peroxisome proliferator activated receptor-gamma (PPAR- γ) [17]. In another study the administration of losartan to children of essential hypertensive parents, with early metabolic abnormalities, was followed by a significant reduction in the levels of TC and TGL [18].

Hence, this study was undertaken to evaluate and compare the safety, efficacy, cardiovascular parameters and pleiotropic effects of Telmisartan and Losartan in patients having mild to moderate hypertension.

2. MATERIALS AND METHODS

Randomized, parallel group, open Label study on the safety and efficacy of Telmisartan and Losartan with their effects on cardiovascular parameters and other pleiotropic effects in mild to moderate hypertension. The total sample size consisted of 100 patients. The inclusion and exclusion criteria was followed by the previous studies [13,18,19]. IEC permission no. 002/SBMC/IHEC/2014-86.

2.1 Study Drugs: 2 Arms

Study group 1: Tab. TELMISARTAN 40 mg once daily (50 patients)

Study group 2: Tab. LOSARTAN 50 mg once daily (50 patients)

Both Telmisartan and losartan were administered orally in tablet form.

2.2 Fasting Lipids

After overnight 12 hours of fasting 5 ml of venous blood sample was collected for estimating Total cholesterol (mg/dl), triglycerides (mg/dl), HDL cholesterol (mg/dl), LDL cholesterol (mg/dl), VLDL cholesterol (mg/dl) using enzymatic method

2.3 Blood Pressure

In resting state sitting blood pressure (mmHg) was recorded with standard sphygmomanometer. Readings were accurately measured at the

interval of 30 minutes and the average was recorded in the data Performa. Patients were advised not to take any beverages, heavy food or strenuous exercise.

2.4 Screening

Screening procedure consisted of a detailed medical and drug history, thorough clinical examination followed by laboratory investigations – which included fasting lipid profile. After screening of 134 patients, 34 patients were excluded based on selection criteria. A total of 100 patients of both sexes and age between 18 to 65 years who fulfilled the selection criteria were recruited for the study

2.5 Randomization

The study subjects were randomly assigned using a computer generated randomization chart to either of the two groups Group A and Group B, each group consisting of 50 patients.

2.6 Group Allocation and Dosage Regimen

Group A: Tab. Telmisartan 40 mg once daily for 6 months.

Group B: Tab. Losartan 50 mg once daily for 6 months.

At the first visit tablets were given for 4 weeks according to the above dosage regimen. Patients were advised to continue antidiabetic medication as before. No special diet instruction was given.

Biochemical investigations such as Fasting Plasma lipid profile, Total Cholesterol, LDL, Triglycerides, VLDL, and HDL.

2.6.1 Primary outcome measure

The primary efficacy end-point was percent change from baseline systolic blood pressure, diastolic blood pressure at the end of Week 24 weeks [Time Frame: Baseline to 24 weeks] .

2.6.2 Secondary outcome measure

Secondary efficacy outcome measures are:

- Percent Change From Baseline in Total Cholesterol (TC) [Time Frame: Baseline to 24 Weeks]

- Percent Change From Baseline in High Density Lipoprotein Cholesterol (HDL-C) [Time Frame: Baseline to 24 weeks]
- LDL [Time Frame: 24 weeks].
- Triglycerides (TG) [Time Frame: 24 weeks].
- TC/HDL-C Ratio [Time Frame: 24 weeks]
- VLDL [Time Frame: 24 weeks]

Secondary safety outcome measures:

Safety evaluation was based on the spontaneously reported adverse events and the changes in the laboratory values after the study.

2.7 Statistical Analysis

Data analysis was performed by means of the SPSS statistical software package for Windows (version 9.0; SPSS Inc., Chicago, USA); results were expressed as the mean±SD. One-way analysis of variance (ANOVA) was used to compare baseline data. Change was calculated as the value obtained at the end of 24 -Week

treatment minus the value obtained at baseline. ANOVA was also used to assess the significance within and between groups. A one-sample Student's-t-test was used to compare values obtained before and after treatment. Values of $p < 0.05$ were considered significant.

3. RESULTS AND DISCUSSION

The overall results of this study are as shown below.

3.1 Matching of the Two Groups

The two groups were matched in respect of their age, cardiovascular parameters and lipid profile before starting the study.

The number of males and females of the two groups were matched in the above Table 7. The improvements of the drug from baseline to 3 month and 6 month were compared between the groups to identify the superiority of the drug in the improvement of the cardiovascular parameters and lipid profile.

Table 1. Age distribution

Variable	Group 1 (TELMISARTAN) n= 50		Group 2 (LOSARTAN) n=50	
	Mean ± SD		Mean ± SD	
Age(years)	46.46 ±5.26		47.44 ±6.27	
SBP	151.72±6.85		150.12±7.72	
DBP	90.92±5.90		91.12±5.67	
HR	88.14±7.99		88.84±7.52	
TC	196.4 ± 17.63		195.7±20.26	
LDL	131.07 ± 18.61		128.66 ± 20.22	
HDL	39.44 ± 6.76		41.76 ± 7.16	
TG	129.46 ± 10.98		126.42 ± 11.48	
VLDL	25.89 ±2.20		25.28± 2.30	
Total CH:HDL	5.16±1.23		4.82±0.92	

Table 2. Gender distribution

GENDER	Group					
	Telmisartan		Losartan		Total	
	Number	%	Number	%	Number	%
Male	22	44%	24	48%	46	46%
Female	28	56%	26	52%	54	54%
Total	50	100%	50	100%	100	100.0

Table 3. Effect of telmisartan on systolic and diastolic blood pressure

	Baseline (Mean±SD)	3 Months (Mean ±SD)	6 Months
SBP(mm Hg)	151.72±6.84	131.62±6.40**	129.98 ±6.12**
DBP(mm Hg)	90.92±5.90	84.92±3.06**	82.72±2.20**

The effect of Telmisartan on Systolic blood pressure during Baseline evaluation was 151.72 \pm 6.84. After 3months of treatment, the systolic BP reduced to 131.62 \pm 6.40The reduction is statistically very highly significant (P<0.001). At the end of the study, after 6months of treatment, the effect on systolic BP has significantly reduced to 129.98 \pm 6.12The reduction is statistically very highly significant (P<0.001).

The effect of Telmisartan on Diastolic blood pressure during Baseline evaluation was 90.92 \pm 5.90. After 3months, diastolic BP reduced to 84.92 \pm 3.06The reduction is statistically very highly significant (P<0.01). At 6months the effect on diastolic BP was significantly reduced to 82.72 \pm 2.20. The reduction is statistically very highly significant (P<0.01). *p<0.05 vs. baseline. **p<0.01 vs. baseline, ns P>0.05-not significant.

The effect of Losartan on Systolic blood pressure during Baseline evaluation was 150.12 \pm 7.72. At 3months, systolic BP reduced to 131.14 \pm 6.13The reduction is statistically very highly significant (P<0.01). At 6months the effect on systolic BP significantly reduced to 128.16 \pm 3.83The reduction is statistically very highly significant (P<0.01).

The effect of Losartan on Diastolic blood pressure during Baseline evaluation was 91.12 \pm 5.67. At 3months, diastolic BP reduced to 84.64 \pm 3.09The reduction is statistically very highly significant (P<0.01). At 6months the effect on diastolic BP significantly reduced to 83.44 \pm 2.56The reduction is statistically very highly significant (P<0.01). *p<0.05 vs. baseline. **p<0.01 vs. baselines P>0.05-not significant.

The effect of Telmisartan on Heart Rate recorded at baseline was 88.14 \pm 7.99. At 3months, heart rate reduced to 85.16 \pm 9.85The reduction is statistically very highly significant (P<0.01). At 6months the effect on heart rate significantly reduced to 83.88 \pm 8.41. The reduction is statistically very highly significant (P<0.01). *p<0.05 vs. baseline. **p<0.01 vs. baseline, nsP>0.05-not significant.

The effect of Telmisartan on Heart Rate recorded at baseline was 88.44 \pm 7.52. At 3months, heart rate reduced to 86.08 \pm 9.34The reduction is statistically significant (P<0.05). At 6months, the effect on heart rate significantly.

Reduced to 84.68 \pm 9.48The reduction is statistically very highly significant (P<0.01). *p<0.05 vs. baseline. **p<0.01 vs. baseline, NS P>0.05-not significant.

Table 4. Effect of losartan on systolic and diastolic blood pressure

	Baseline	3 Months	6 Months
SBP (mm Hg)	150.12 \pm 7.72	131.14 \pm 6.13**	128.16 \pm 3.83**
DBP(mm Hg)	91.12 \pm 5.67	84.64 \pm 3.09**	83.44 \pm 2.56**

Table 5. Effect of telmisartan on heart rate

	Baseline	3 Months	6 Months
HEART RATE (PULSE/MIN)	88.14 \pm 7.99	85.16 \pm 9.85**	83.88 \pm 8.41**

Table 6. Effect of losartan on heart rate

	Baseline	3 Months	6 Months
HEART RATE (PULSE/MIN)	88.44 \pm 7.52	86.08 \pm 9.34*	84.68 \pm 9.48**

Table 7. Effect of telmisartan on lipid profile

	Baseline	3 Months	6 Months
TC(mg/dl)	196.40 \pm 17.63	193.12 \pm 15.81*	188.22 \pm 13.19**
LDL-C(mg/dl)	131.06 \pm 18.61	126.43 \pm 17.20*	120.73 \pm 14.63**
HDL-C(mg/dl)	39.44 \pm 6.75	41.12 \pm 5.43*	42.12 \pm 5.10**
TG(mg/dl)	129.46 \pm 10.97	127.82 \pm 10.21 ^{ns}	126.84 \pm 9.60*
VLDL(mg/dl)	25.89 \pm 2.19	25.56 \pm 2.04 ^{ns}	25.36 \pm 1.92*

The effect of Telmisartan on Total cholesterol at baseline evaluation was 196.40±17.63, which reduced marginally at 3months evaluation 193.12±15.81The reduction is statistically very highly significant (P<0.05). At 6 months total cholesterol level significantly reduced to 188.22±13.19The reduction is statistically very highly significant (P<0.01).

The effect of Telmisartan on LDL-C recorded at baseline was 131.06±18.61, at 3months decreased to 126.43±17.20The reduction is statistically significant (P<0.05). At 6 months the LDL-C value significantly reduced to 120.73±14.62. The reduction is statistically very highly significant (P<0.01).

The effect of Telmisartan on HDL-C at baseline evaluation was 39.44±6.75,at 3months value increased to 41.12±5.43The improvement is statistically very highly significant (P<0.05). At 6months, HDL-C significantly increased to 42.12±5.10The improvement is statistically very highly significant (P<0.01).

The effect of Telmisartan on Triglycerides level at baseline evaluation was 129.46±10.97, which reduced marginally to 127.82±10.21 at 3monthsThe reduction is statistically not significant (P>0.05). At 6 months, Triglycerides level was further reduced to 126.84±9.60 the reduction is statistically significant (P<0.05).

The effect of Telmisartan on VLDL level at baseline evaluation was 25.89±2.19. At 3months the value reduced marginally to 25.56±2.04The reduction is statistically not significant (P>0.05). At 6 months, the VLDL level was 25.36±1.92 The reduction is statistically significant (P<0.05). *p<0.05 vs. baseline. **p<0.01 vs. baseline, ns P>0.05-not significant.

The effect of Losartan on Total cholesterol recorded at baseline evaluation was 195.70±20.26, which reduced marginally at 3 months evaluation 194.86±21.64The reduction is statistically not significant (P>0.05). At 6months, total cholesterol level was 192.94±19.00The reduction is statistically significant (P<0.05).

The effect of Losartan on LDL-C recorded at baseline evaluation was 128.65±20.21; at 3months decreased to 128.88±17.20The reduction is statistically not significant (P>0.05). At 6months the LDL-C value was 126.97±19.05The reduction is statistically not significant (P>0.05).

The effect of Losartan on HDL-C at baseline evaluation was 41.76±7.16,at 3months value decreased to 40.80±6.04The reduction is statistically not significant (P<0.05). At 6 months, HDL-C was 41.04±6.08The improvement is statistically not significant (P>0.05).

The effect of Losartan on Triglycerides level at baseline evaluation was 126.42±11.47 which reduced marginally to 125.90±7.95 at 3 monthsThe reduction is statistically not significant (P>0.05). At 6months, the Triglycerides level was 124.64±10.70 the reduction is statistically not significant (P>0.05).

The effect of Losartan on VLDL level at baseline evaluation was 25.28±2.29. At 3 months the value reduced marginally to 23.18±1.59The reduction is statistically not significant (P>0.05). At 6months, VLDL level was 24.92± 2.14The reduction is statistically not significant (P>0.05).

*p<0.05 vs. baseline. **p<0.01 vs. baseline, nsP>0.05-not significant.

3.2 Total Cholesterol: HDL-Cholesterol Ratio

The Effect of Telmisartan on Total cholesterol-HDL cholesterol ratio recorded at baseline was 5.16. At the end of 3 rd months, the ratio has reduced to 4.79, and Total cholesterol-HDL cholesterol ratio further reduced to 4.54 at the end of 6 months.

The Effect of Losartan on Total Cholesterol-HDL cholesterol ratio recorded at baseline was 4.82. At the end of 3 rd. month it has increased to 4.87, and Total Cholesterol-HDL cholesterol ratio slightly reduced to 4.80 at the end of 6 months.

Table 8. Effect of losartan on lipid profile

	Baseline	3 Months	6 Months
TC(mg/dl)	195.70±20.26	194.86±21.64 ^{ns}	192.94±19.00*
LDL-C(mg/dl)	128.65±20.21	128.88±17.20 ^{ns}	126.97±19.05 ^{ns}
HDL-C(mg/dl)	41.76±7.16	40.80±6.04 ^{ns}	41.04±6.08 ^{ns}
TG(mg/dl)	126.42±11.47	125.90±7.95 ^{ns}	124.64±10.70 ^{ns}
VLDL(mg/dl)	25.28±2.29	23.18±1.59 ^{ns}	24.92±2.14 ^{ns}

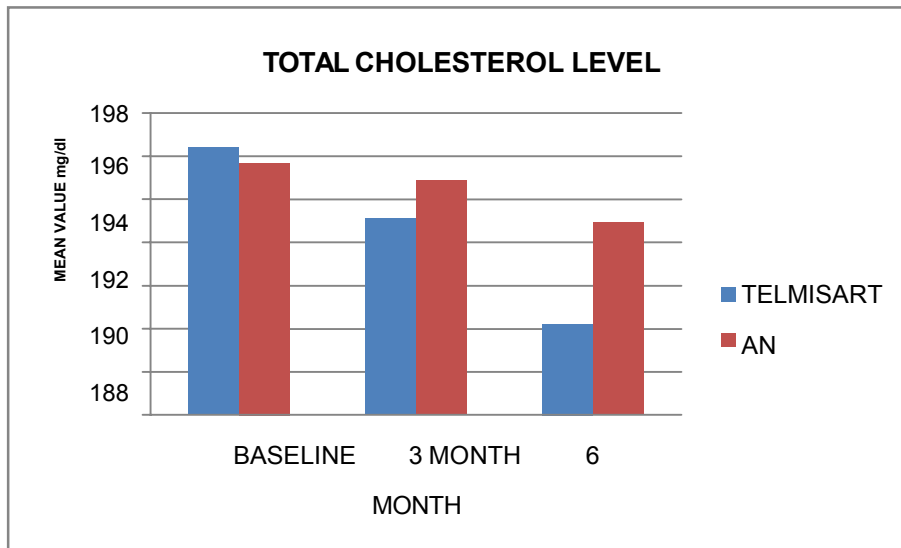


Fig. 1. Effect of telmisartan and losartan on total cholesterol

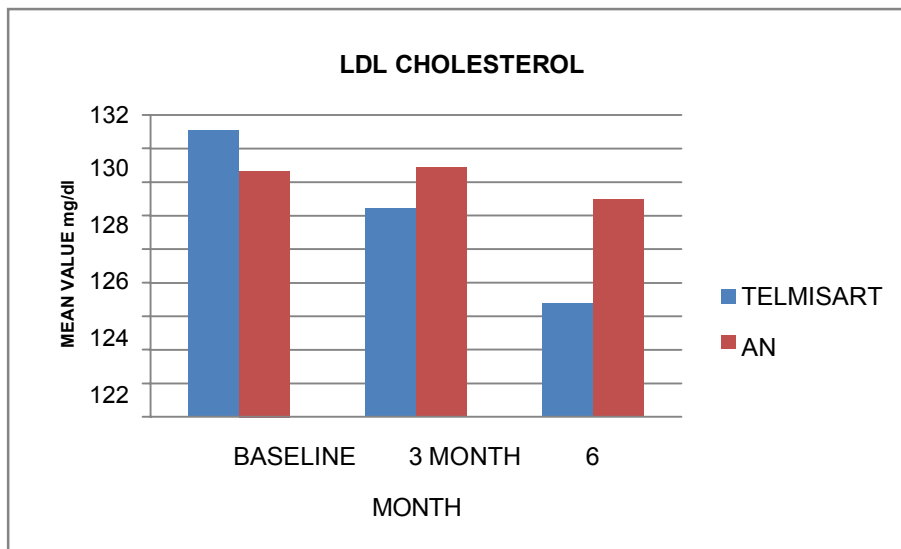


Fig. 2. Effect of telmisartan and Losartan on LDL cholesterol

3.3 Blood Pressure Control

After 6 months of treatment, both telmisartan and losartan significantly ($p < 0.01$) reduced SBP compared with baseline (Table 8 and 9). There was also a significant ($p < 0.01$) decrease in DBP in telmisartan-treated patients compared with baseline. Mild significant changes in heart rate compared with baseline were observed in both of the treatment groups.

The importance of aggressive blood pressure control in patients with type 2 diabetes in

reducing cardiovascular complications has been demonstrated by the UK Prospective Diabetes Survey [3]. In patients achieving a blood pressure of 144/82 mmHg, risks of heart failure, stroke is less. The Hypertension Optimal Treatment study subsequently showed that intensive blood pressure control to 130/85 mmHg by the use of a combination of antihypertensive drugs resulted in further risk reduction (96).

3.4 Lipid Profile

After 6-month telmisartan treatment, there was a significant reduction in plasma TC (Table 8). At 6

months, telmisartan treatment resulted in a significant reduction in plasma TC ($p < 0.01$), LDL-C ($p < 0.01$), VLDL ($p < 0.05$) and TG ($p < 0.05$) and increment in HDL ($p < 0.01$) value compared with baseline. In losartan mild significant changes ($TC < 0.05$) compared with baseline in the lipid profile of patients who had received losartan for 6 months, there is no significant changes in other parameters. The reductions in lipid levels achieved with telmisartan were significantly greater ($p < 0.05$) than those by Losartan.

In patients with hypertension, the treatment aim is to control hypertension and maintain a favorable lipid profile in order to minimize

cardiovascular complications and improve patient prognosis.

Nevertheless, pharmacological intervention may be necessary to control blood pressure and minimize cardiovascular complications. The importance of angiotensin II in the pathophysiology of cardiovascular disease is well known. Recently, the relationship between cardiovascular disease, diabetes and hypertension and the role of peroxisome proliferator-activated receptor- γ (PPAR γ) has been demonstrated. Preliminary data suggest that the ARB telmisartan increases PPAR γ activity and may provide additional benefits in the treatment of hypertension [20,21].

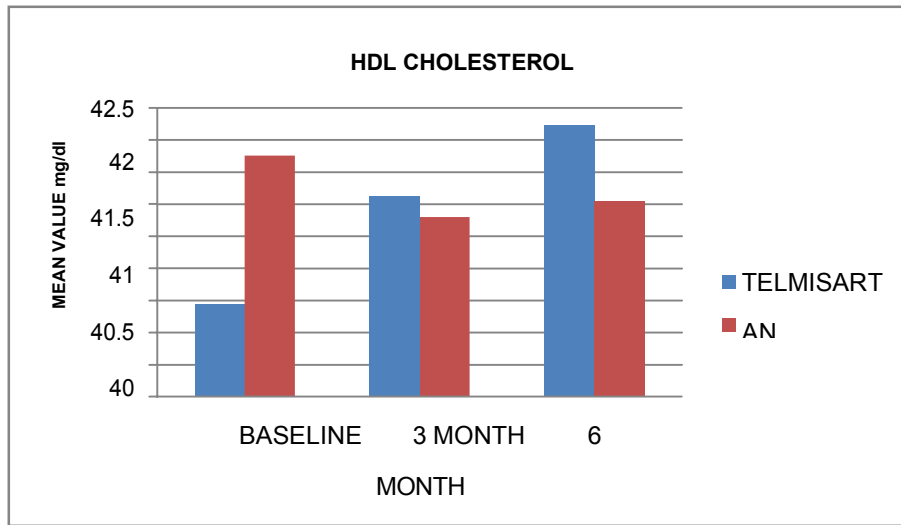


Fig. 3. Effect of telmisartan and losartan on hdl cholesterol

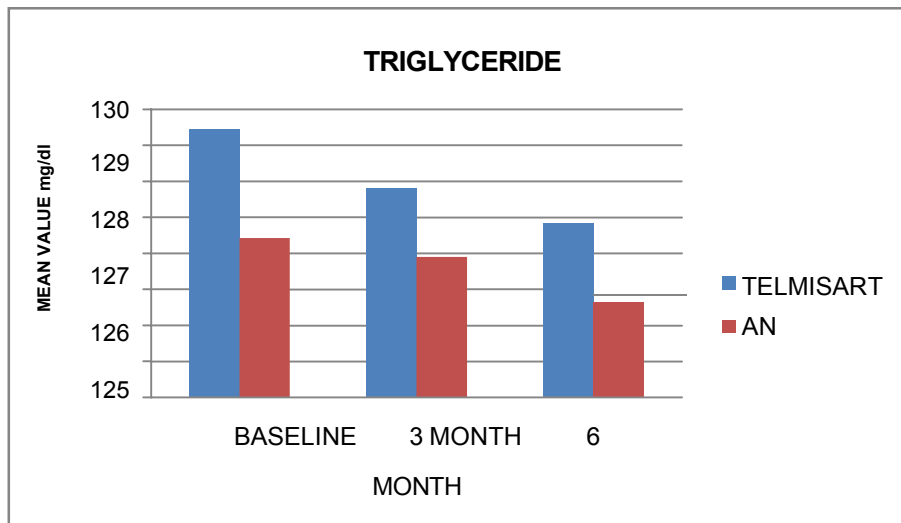


Fig. 4. Effect of telmisartan and losartan on triglyceride

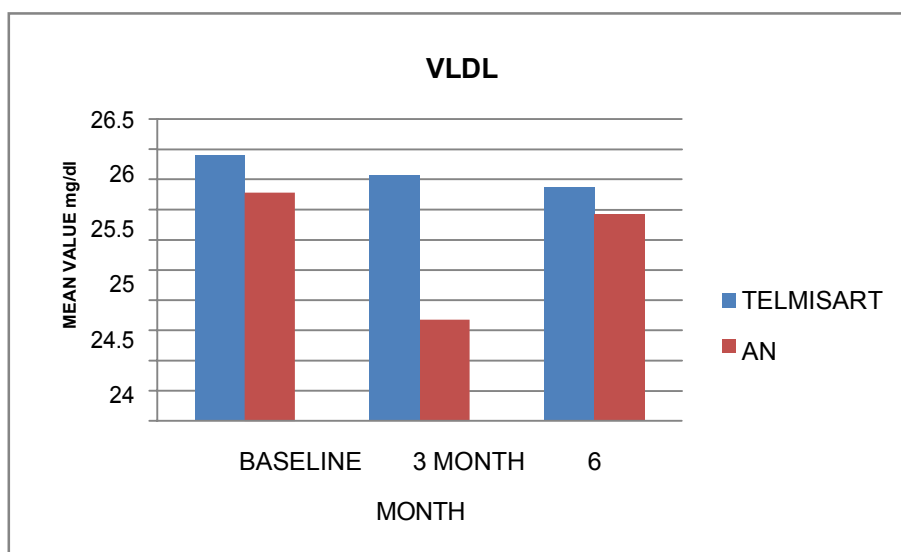


Fig. 5. Effect of telmisartan and losartan on VLDL cholesterol

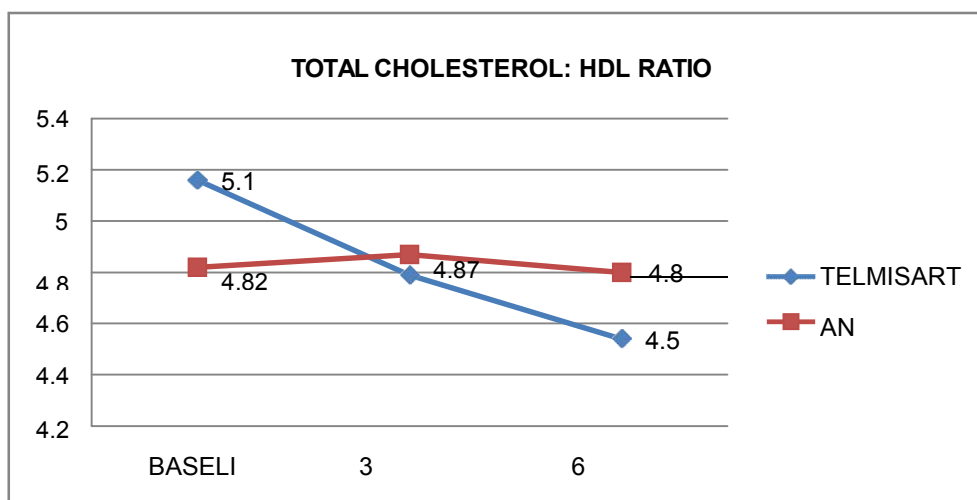


Fig. 6. Effect of telmisartan and losartan on total cholesterol: HDL cholesterol ratio

Table 9. Incidence of adverse effects

S. No	Adverse effect	Telmisartan Group (50)	Losartan group (50)
1	Nausea	-	-
2	Fatigue	1(2%)	1(2%)
3	Dizziness	1(2%)	2(4%)
4	Head ache	2(4%)	3(6%)
5	Skin rash	-	-
	Total	4	6

In addition, the beneficial effects of a low dose of an ARB in the prevention of target organ damage have been demonstrated in mild to moderate hypertensive patients with prevention of early

kidney damage being independent of the antihypertensive activity [22]. The term "pleiotropic" borrowed by the author from the terminology of genetics, in the best possible way

reflects the nature of the biologically active compounds (BACs) and medicines in complex biological systems and in vivo, organized by certain biological (pharmacological) effect induction. In genetics pleiotropic refers to the impact of a single gene in the development of several phenotypic traits. In this context, pleiotropic means the ability of the BACs and medicines to implement more than one mechanism of action resulting in the specific biological (pharmacological) effect. The interaction of these mechanisms forms a distinct pattern of biological response (pleiotropic pattern), which reflects the change in his Character with the increased dose (concentration)-dependent efficacy of BACs and medicines [22]. The advantages of the longer action of telmisartan may extend to the prevention of cardiovascular events, which follow a similar circadian pattern, with an increase in their incidence being associated with the early morning blood pressure surge. For a drug with a short half-life taken in the morning, the time of maximal cardiovascular risk is likely to coincide with a period of suboptimal blood pressure control [23-25].

The plasma lipid profile of hypertension patients is especially important given because of high incidence of atherosclerosis. In comparison with once-daily losartan 50 mg, telmisartan treatment at a dose of 40 mg once daily resulted in a significant reduction in plasma LDL-C, TC and triglyceride and increase of HDL level. With losartan end investigation of lipid levels were similar to baseline investigation. This distinction between telmisartan and losartan may possibly be explained by the high lipophilicity of telmisartan compared with losartan and other ARBs and the PPAR γ -modulating effect of telmisartan. Other Structural differences among the ARBs may also contribute to the possible differences in metabolic profiles [19,26]. In contrast, PPAR α is highly expressed in tissues displaying a high metabolic rate of fatty acids, such as the liver and skeletal muscle. PPAR α modulates intracellular lipid metabolism by transcriptional regulation of genes involved in fatty acid uptake, mitochondrial fatty acid oxidation and triglycerides metabolism. PPAR α is the molecular target of fibrates such as Gemfibrozil, etc. Thus there is induction of hepatic ACSL1 (acyl CoA synthetase long chain) and CPT1A (carnitine palmitoyl transferase). This causes significant decrease of triglyceride level. PPAR α in skeletal muscle is not affected by Telmisartan. Hence the myopathy associated

with fibrates is not seen with Telmisartan. Thus PPAR α . Activation by Telmisartan is liver specific because of its specific pharmacokinetic profile [25]. Telmisartan has also been approved for the primary prevention of stroke and prevention of MI. It decreases the cerebral infarct area in dose dependent manner without affecting cerebral blood flow. Thus it could be a potential target for the treatment of post – ischemic injury by partially inhibiting the inflammatory reaction after cerebral ischemia via a PPAR – gamma dependent mechanism [19,26]. The multimodal mechanism of action of Telmisartan, including AT1 receptor blockade, PPAR γ modulation and hepatic PPAR α activation, characterizes this compound as a therapeutic option for the treatment of patients suffering from multiple cardio metabolic disorders such as hypertension, glucose intolerance, and dyslipidemia [27,28].

4. LIMITATIONS

- Small sample size
- Shorter duration of study
- Both of the drugs had showed epithelial dysfunction as their pleiotropic
- Effects in previous studies [23,24].

5. CONCLUSION

These results suggest that there are differences between ARBs. Telmisartan, after administration for 6 months, conferred significant advantages compared with losartan in terms of blood pressure control and plasma lipid levels with hypertension. The antihypertensive treatment with Telmisartan may result in an amelioration of cardiovascular risk factors, not only through arterial pressure regulation but also through the reduction of lipid markers. No data are available on the relationship between ARB and 3-hydroxy-3-methylglutaryl- coenzyme A (HMG- CoA) reductase inhibition. It is possible that telmisartan, because of its long duration of action, may have additive effects on the inhibition of HMGCoA reductase. Further studies are required to assess such possibilities.

In conclusion, a wide number of drugs are available today for effective treatment of dyslipidemia. The pleiotropic effects of statins offer advantages over other available lipid lowering agents, as they are effective, well tolerated and can provide additional benefits not only in cardiovascular disorders but in other diseases too.

CONSENT AND ETHICAL APPROVAL

As per international standard or university standard guideline participant consent and ethical approval has been collected and preserved by the authors.

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COMPETING INTERESTS

Authors have declared that no competing interests exist.

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